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- No letter should be more than 400 words.
- For letters on scientific subjects we normally reserve our correspondence columns for those relating to issues discussed recently (within six weeks) in the *BMJ*.
- We do not routinely acknowledge letters. Please send a stamped addressed envelope if you would like an acknowledgment.
- Because we receive many more letters than we can publish we may shorten those we do print, particularly when we receive several on the same subject.

Hospital emergency numbers

After recent alterations to hospital switchboard equipment this hospital was obliged to change its internal emergency number (the number dialled to summon the cardiac arrest team). The change from 666 to 300, although reasonably well publicised, generated some confusion among users. The telephonists also were unhappy and regarded this new number, which had been selected by the hospital engineer, as unsuitable.

To find what other numbers were in use and whether such a change had occurred elsewhere I contacted 72 hospitals in the north of England. I found that there were 22 different emergency numbers and that about a fifth of these had been changed during the past five years. The number 222 was the most popular choice and was found in about a quarter of the hospitals surveyed. Most were simple numbers, being composed of a single digit repeated three or four times (usually two, three, four, five, or six and occasionally eight and nine). Some numbers were more complex, being either progressions—for example, 123, 234, and 345—or symmetrical—for example, 212, 313, 515, and 616. In about a 10th of hospitals semirandom sequences had been adopted—for example, 311, 299, and even 3511). There was minimal uniformity on either a regional or district basis. One city with five hospitals all within the same district

health authority had five different emergency numbers. Replacement of switchboard equipment was the only reason given for change of these numbers. Surprisingly, only one hospital had used 999 as its emergency number despite its universal acceptance outside hospital.

Obviously, differing and changing emergency numbers are most unsatisfactory. Several factors need to be taken into account when selecting an internal emergency number for a hospital. Most of the staff using the number do so infrequently, and many of them work or have worked in several hospitals—for example, staff in training and agency staff. The number is often dialled in an atmosphere of some panic, when reason, memory, and coordination may be impaired. The number should therefore be constant, easy to dial, unforgettable, and obvious. The ideal choice for use both inside and outside hospital is 999. A safe and easy way of implementing this change would be to add 999 as an internal emergency number to every hospital switchboard. The existing emergency numbers could remain operative until they fell into disuse.

Advances in communication technology should surely be exploited to simplify rather than to confuse emergency procedures.

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Coronary flow reserve and the J curve

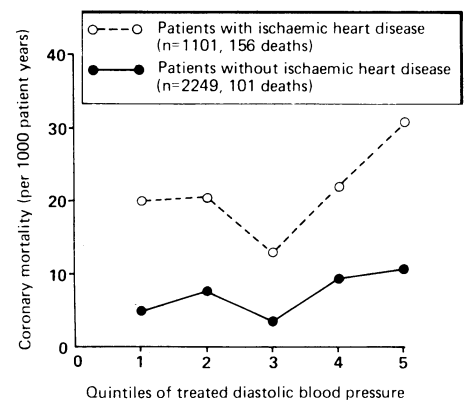
Dr John M Cruickshank (12 November, p 1227) has been persistent in his contention that lowering blood pressure too far may be harmful,^{1,4} but his hypothesis is only one explanation for the available evidence. The indifferent quality of the data was highlighted by Dr D G Beevers (12 November, p 1212) in his accompanying editorial. None of the studies cited in the table on page 1227 allows strong inferences about causality, and in most the number of deaths from myocardial infarction was small. The reanalysis of data from Clatterbridge (fig 4, p 1229) was based on a total of 25 deaths.¹ It seems that four out of nine points on the graph are each determined by a single death. Furthermore, from 1972 to 1976 large doses of atenolol (up to 1200 mg daily and occasionally even higher) were used in the Clatterbridge clinic.² The mortality during this period was four times greater than during 1977-82, when much lower doses (100-200 mg daily) were used.² The possible implications of this observation have yet to be addressed.

The apparent consistency in juxtaposing the findings of several studies (fig 1, p 1228) is misleading. Some of the data relate only to patients with pre-existing ischaemic heart disease¹ and

some to allcomers,⁵ while two studies excluded patients with previous myocardial infarction altogether.^{6,7} As Dr Beevers points out, to sustain his hypothesis Dr Cruickshank has to ignore findings of J curves in patients without ischaemic heart disease.^{6,7} The Glasgow study⁸ had a total of 257 deaths and clearly showed a non-linear relation between diastolic blood pressure and death from myocardial infarction, regardless of whether ischaemic heart disease was present at entry (figure).

Although we favour a different interpretation of the J curve phenomenon,³ the fundamental issue for the practising doctor is whether the data are sufficiently compelling to warrant a change in treatment practice—that is, to avoid lowering diastolic blood pressure below 85 mmHg in patients who may have ischaemic heart disease. Those who argue for this policy are doing so without first evaluating its feasibility (can blood pressure really be controlled to within 5 mmHg?), efficacy (will coronary deaths be prevented without increasing the stroke rate?), or resource implications (would more visits to the surgery or clinic be necessary?).

We believe that no alteration of practice is justified, but purpose designed studies should be performed to determine the ideal blood pressure during treatment. In Sweden a randomised study



Coronary mortality in Glasgow Blood Pressure Clinic by quintiles of achieved diastolic blood pressure for patients with and without ischaemic heart disease at entry

designed to examine the effect of intensifying the treatment of patients with a diastolic blood pressure in the range 90-100 mmHg has already started.⁴ Surprisingly, citing Cruickshank's data,¹ the investigators have excluded patients with ischaemic heart disease on ethical grounds. Sadly therefore, their study will not confirm or refute the hypothesis in question. Further speculation on the J curve phenomenon will be unproductive, the priority should be to collect some adequate evidence.

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- 1 Cruickshank JM, Thorp JM, Zacharias FJ. Benefits and potential harm of lowering high blood pressure. *Lancet* 1987;351:581-3.
- 2 Cruickshank JM, Pennert K, Sorman AE, Thorp JM, Zacharias FM, Zacharias FJ. Low mortality from all causes, including myocardial infarction, in well-controlled hypertensives treated with a beta-blocker plus other antihypertensives. *J Hypertens* 1987;5:489-98.
- 3 Cruickshank JM, Higgins TJ, Pennert K, Thorp JM, Zacharias FM, Zacharias FJ. The efficacy and tolerability of anti-hypertensive treatment based on atenolol in prevention of stroke and the regression of left ventricular hypertrophy. *Journal of Human Hypertension* 1987;1:87-93.
- 4 Cruickshank JM. Why has primary prevention of myocardial infarction in the treatment of hypertension been so elusive? *Journal of Human Hypertension* 1987;1:73-81.
- 5 Waller PC, Isles CG, Lever AF, Murray GD, McInnes GT. Does therapeutic reduction of diastolic blood pressure cause death from ischaemic heart disease? *Journal of Human Hypertension* 1988;2:7-10.
- 6 Wilhelmsson L, Berglund G, Elmfeldt D, et al. Beta-blockers versus diuretics in hypertensive men: main results from the HAPPHY trial. *J Hypertens* 1987;5:561-72.
- 7 Samuelsson O, Wilhelmsson L, Andersson OK, Pennert K, Berglund G. Cardiovascular morbidity in relation to change in blood pressure and serum cholesterol levels in treated hypertension: results from the primary prevention trial in Göteborg, Sweden. *JAMA* 1987;258:1768-76.
- 8 The BBB Study Group. The BBB study: a prospective randomised study of intensified antihypertensive treatment. *J Hypertens* 1988;6:693-7.